General Principles of Mechanical Ventilation

9

General Principles of Mechanical Ventilation

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Chapter Outline

HISTORY

MECHANICAL VENTILATION

Basic Concepts

The Ventilatory Circuit

Alarms and Safety

Automatic Tube Compensation

INDICATIONS FOR MECHANICAL VENTILATION

MECHANICAL BREATH GENERATION

VENTILATOR MODES

Continuous Mandatory Ventilation

Assist Control Ventilation

Synchronized Intermittent Mandatory Ventilation

Stand-Alone Pressure Support Ventilation

Dual Control Modes

OTHER MODES OF MECHANICAL VENTILATION

Airway Pressure Release Ventilation

High-Frequency Oscillatory Ventilation

Effort-Adapted Modes of Mechanical Ventilation

POSITIVE END-EXPIRATORY PRESSURE

MONITORING THE VENTILATED PATIENT

Hemodynamics

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Ventilator Waveforms

MAINTAINING SUPPORT OF THE VENTILATED PATIENT

Sedation and Analgesia

Positioning

Secretion Clearance

WEANING FROM MECHANICAL VENTILATION

COMPLICATIONS OF MECHANICAL VENTILATION

Hemodynamics

Ventilator-Induced Lung Injury

Ventilator-Associated Pneumonia

Gastrointestinal Bleeding

Venous Thromboembolism

NONINVASIVE POSITIVE-PRESSURE VENTILATION

Management of the mechanically ventilated patient is a cornerstone of critical care training and practice. The institution of mechanical ventilation can be a lifesaving measure. However, the mechanical ventilator also has potential for great harm and, in and of itself, does not reverse underlying disease. Limiting iatrogenic injury from ventilator-induced lung injury (VILI) should take high priority, along with acceptable levels of oxygenation and ventilation. The clinician should be aware of basic and advanced principles involving mechanical ventilation, allowing flexibility when applying evidence-based practices to the individual patient. Knowledge of guidelines and large clinical trials is vitally important, and the consideration of patient trajectory, individual physiology, timing of therapy, and severity of illness will make tailoring the ventilator prescription most effective.[1]

History

Hippocrates (460-370 BC) likely gave the first description of endotracheal intubation in his “Treatise on Air,” in which he states that “One should introduce a cannula into the trachea along the jawbone so that air can be drawn into the lungs.”[2] In 1530, Paracelsus (1493-1541) used a fire bellows connected to a tube inserted into a patient’s mouth as a ventilator device.[3] The first known mechanical device designed specifically to provide ventilation for the patient was the foot pump developed by Fell and O’Dwyer in the 1880s.[4]

The first generation of mechanical ventilators focused primarily on the intermittent delivery of a bulk volume of gas to the patient with limited monitoring.[5] Negative-pressure ventilators were invented and applied a negative pressure around the body or chest cavity. Two classic devices that provided negative-pressure ventilation were the iron lung and the chest cuirass or chest shell.[6] Iron lungs were widely used during the poliomyelitis epidemics of the 1930s and 1940s. These devices encased the patient from the neck down and applied negative pressure around the patient to expand the lungs. The chest cuirass was intended to alleviate the problems of patient access and “tank shock” that occurred secondary to venous pooling during the application of negative pressure associated with iron lungs.[7] Although the chest cuirass improved patient access and decreased the potential for tank shock, ventilation with this device was limited by the difficulties in [illegible] seal between the shell and the patient’s chest wall.

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[illegible]ic of the 1960s, the era of respiratory intensive care emerged, as positive-pressure ventilation via an artificial airway became commonplace.[8] Controlled mechanical ventilation eventually led to

assisted modes of support, and positive end-expiratory pressure (PEEP) was introduced in the late 1960s. The improvements in mechanical ventilators came about as understanding was gained in manipulating variables of flow and pressure for patient benefit. Further technical evolution of ventilators included advances such as intermittent mandatory ventilation, and synchronous intermittent mandatory ventilation. Modern ventilators now boast microprocessors that serve both in the operating mechanism of the device and in the monitoring systems, enabling automatic adjustment of most aspects of the mechanical breath being delivered.

Mechanical Ventilation

Basic Concepts

For a breath to be generated, a pressure gradient must exist. During normal spontaneous breathing, the diaphragm and other respiratory muscles create gas flow by lowering pleural, alveolar, and airway pressures relative to atmospheric pressure. Alveolar pressure is normally atmospheric at end-inspiration and end-expiration. Diaphragmatic and intercostal muscle activation during normal inspiration expands the chest and decreases intrapleural pressure from –5 cm H_2O to –8 cm H_2O. Alveolar pressure fluctuates from +1 cm H_2O during exhalation to –1 cm H_2O during inspiration.

A ventilation/perfusion ($\dot{V}/\dot{Q}$) mismatch occurs when areas of the lung are perfused but either poorly ventilated (low $\dot{V}/\dot{Q}$) or not ventilated at all (shunt). The latter is an intrapulmonary (IP) (capillary) shunt. Shunt may also be intracardiac (anatomic). Venous admixture, as a measure of less than fully oxygenated blood after passing through the lung, includes both low $\dot{V}/\dot{Q}$ areas of lung and IP shunt. Normal venous admixture is about 2% to 5%. Mechanical ventilation may increase the venous admixture to approximately 10% in the normal individual. Mechanical ventilation usually decreases venous admixture in alveolar lung disease, such as acute lung injury (ALI), improving the distribution of ventilation especially in previously underventilated lung areas. Pressures greater than alveolar opening and closing pressures expand the collapsed alveolus and prevent its collapse, respectively. However, if positive-pressure ventilation produces overdistention, redistribution of pulmonary blood flow to unventilated regions may occur, resulting in hypoxemia. *Dead space* refers to areas of the lung with a higher $\dot{V}/\dot{Q}$ ratio. *Anatomic dead space* is the volume of the conducting airways of the lungs, about 150 mL. *Alveolar dead space* refers to alveoli that are overventilated relative to perfusion; it is increased by any condition that reduces pulmonary blood flow, such as pulmonary embolism (PE) or with overdistention of the lung. *Mechanical dead space* refers to the rebreathed volume of the ventilator circuit; this volume behaves like an extension of the anatomic dead space. Mechanical ventilation can also increase dead space if it leads to overdistention.

An increased dead space fraction requires a greater minute ventilation to maintain alveolar ventilation and $PaCO_2$ (partial pressure of carbon dioxide in arterial blood). Hyperventilation lowers $PaCO_2$. Hypoventilation raises $PaCO_2$; a modest elevation (50-70 mm Hg) reduces pH and is usually not by itself injurious in the mechanically ventilated patient. It has become increasingly recognized that hypercapnia during mechanical ventilation is well tolerated and may not be harmful. An exception occurs in the presence of increased intracranial pressure (ICP).

Although modern ventilators have evolved into complex machines, the basic premise remains: a ventilator is designed to replace or augment a patient's muscles in performing the work of breathing.[8] Ventilators use input power (electricity or compressed gas) to ventilate the lungs. To generate a breath (whether it be spontaneous or positive pressure), a pressure gradient must be generated from the airway opening to the alveoli. The volumes delivered and pressures generated largely depend on the mechanical properties of the respiratory system: the lungs and chest wall as well as the abdomen.[8] Each of these components has mechanical properties that determine the overall behavior of the respiratory system. Although the respiratory [illegible] omplex, the main *variables* of interest are pressure, flow, and volume. The ventilator [illegible] *sure* to cause *flow* through an open circuit and therefore increase lung *volume*.[9] The pressure required to do this reflects a combination of the pressures in the lungs and chest wall. This can

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be illustrated by the equation of motion:[9,10]

$$\text{Muscle pressure} + \text{Ventilator pressure} = (\text{Elastance} \times \text{Volume}) + (\text{Resistance} \times \text{Flow})$$

Compliance describes the ease or difficulty of the respiratory system to expand in response to a delivered pressure and volume. Simplistically, compliance is defined by the change in volume (ΔV) divided by the change in pressure (ΔP), and the compliance of the respiratory system (C_{RS}) is $\Delta V/\Delta P_{alveolar}$. *Elastance* is the inverse of compliance, or the ratio of pressure change to volume change, and describes the tendency to recoil. *Resistance* describes the impedance to airflow through the respiratory system, or the ratio of pressure change to flow change. The *elastic load* is the pressure required to overcome the elastance of the respiratory system, and the *resistive load* is the pressure required to overcome flow resistance of the ventilator circuit, endotracheal tube, and airways.[8]

The equation of motion illustrates several basic principles. To drive gas into the patient, a ventilator can directly control either the pressure or the flow and volume applied at the airway. Despite its complexity a ventilator is simply a machine controlling one of these two variables (*control* variables). For example, in pressure control and pressure support ventilation (PSV), pressure is the control variable and is held constant, although flow and lung inflation will vary based on the patient's respiratory mechanics and inspiratory time (I time) (the latter set directly in pressure control and patient influence with pressure support). In volume ventilation, flow is the control variable and pressure and lung inflation vary with the patient's respiratory mechanics. Flow and volume are intimately linked: volume (L) = flow (L/second) × I time (second).

The Ventilator Circuit

The ventilator circuit consists of plastic tubing connecting the artificial airway or mask with the mechanical ventilator. Within the circuit may reside humidifiers, devices for the delivery of aerosolized medications, filters, suction catheters for secretion clearance, and heated wires.[11] The length and compliance (2-3 cm^3/cm H_2O) of the ventilator circuit are responsible for a volume of gas contained within the circuit, termed the *compressible volume*. This is partly responsible for the discrepancy between the set tidal volume delivered and the expiratory volume measured and displayed by the ventilator. The ventilator circuit also adds resistance to the system, but is minimal when compared to either the patient's inherent mechanics or the endotracheal tube. Although frequently colonized with bacterial pathogens, the routine change of the ventilator circuit for infection prevention (e.g., ventilator-associated pneumonia) is not recommended.

In the body, inspired gases are conditioned in the airway just before the carina so that they are fully saturated with water at body temperature by the time they reach the alveoli (37° C, 100% relative humidity, 44 mg/L absolute humidity, 47 mm Hg water vapor pressure). This portion of the airway acts as a heat and moisture exchanger (HME). Under normal conditions, about 250 mL of water is lost from the lungs each day to humidify the inspired gases.

Gases delivered from mechanical ventilators are typically dry, and the upper airways of patients being ventilated are functionally bypassed by artificial airways, necessitating the use of an external humidifying apparatus in the breathing circuit. Because the upper airway is bypassed during mechanical ventilation, the inspired gas temperature should be kept close to the body temperature. Inspired gases that bypass the upper respiratory tract through endotracheal tubes or tracheostomy tubes should be heated to at least 32° to 34° C at 95% to 100% relative humidity. The temperature probe for the heated humidifier should be placed inside the inspiratory limb of the ventilator circuit as close to the patient as possible. Moisture loss and subsequent dehydration of the respiratory tract result in epithelial damage.

An HME, which is placed between the artificial airway and the ventilator circuit, may be used to replace the traditional heated humidifier. During exhalation, moisture and heat from the patient are absorbed into the [illegible] of the exchanger and are transferred back to the patient during the next inhalation. [illegible] bacterial-viral filtering HMEs cost less to maintain and are less likely to colonize [illegible] with heated humidifiers.[12,13] Contraindications for use of an HME are thick or large amounts of secretions, minute volume exceeding 10 L/minute, body temperature less than 32° C, and need

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for aerosolized medications.[14]

Alarms and Safety

Current ventilators are equipped with monitors that constantly or periodically assess the ventilator's operation and the patient's status (Fig. 9.1). These monitors are usually associated with alarms that visually or audibly notify the operator of any variation from the preset norm. Ventilator alarms can warn of potentially life-threatening events, must have an appropriate level of sensitivity and specificity, and must be evaluated clinically and in a clinical context.[15] There are some alarms related to *power input* (e.g., low battery, loss of power, loss of air supply) and *control circuit* (e.g., nonfunctioning ventilator, incompatible settings), but most alarms are related to measured *output*, such as pressure, volume, and flow.[8]

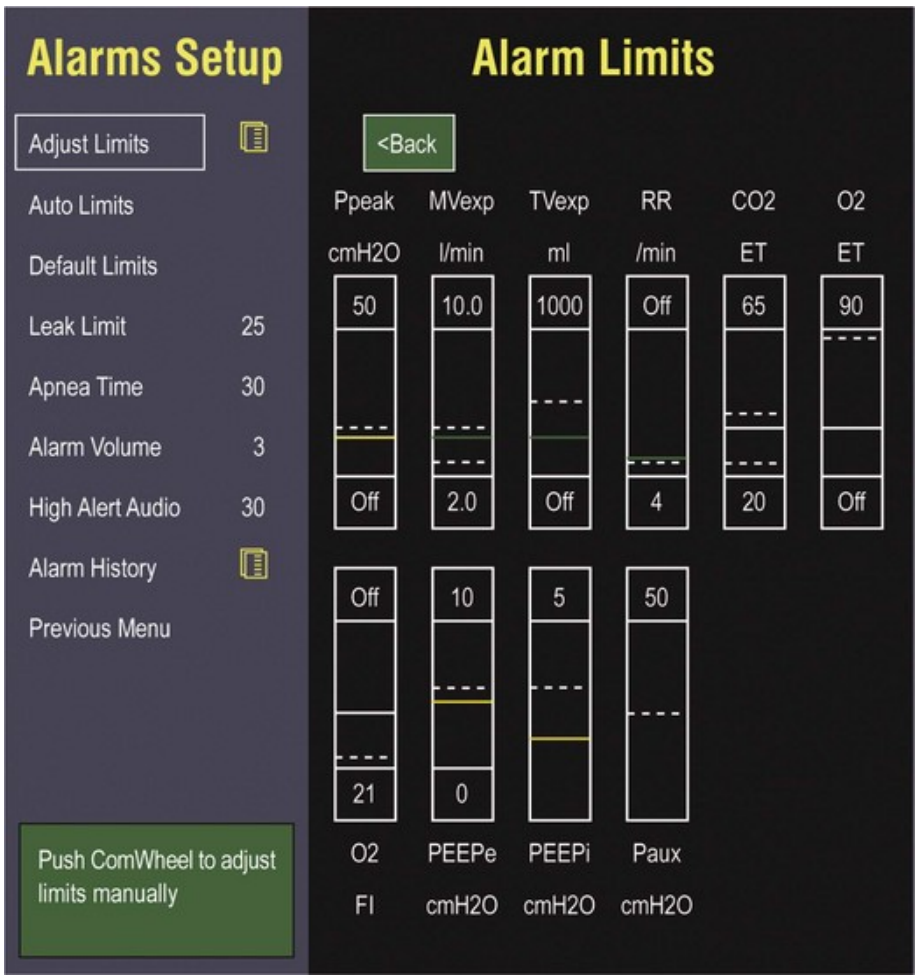

Figure 9.1 The alarm panel of a Draeger Evita 4 ventilator showing the alarms typically used on current ventilators. (Reproduced with permission from Draeger Medical, Inc., Lübeck, Germany.)

High-pressure alarms are triggered by patient factors (such as decreased compliance and increased resistance of the respiratory system) or by ventilator circuit malfunction (obstruction or kinking of the endotracheal tube). Low-pressure alarms are generally secondary to a leak in the system (ventilator circuit, endotracheal tube or cuff) or patient (large pressure loss from a bronchopleural fistula). A high expired volume alarm could be seen with improved pulmonary mechanics during pressure-control ventilation. Low expired volume could be secondary to patient-ventilator disconnect or a leak in the system or patient. A high-frequency alarm is secondary to either autotriggering or hyperventilation, and a low-frequency alarm indicates bradypnea or apnea.[8,15]

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ompensation

Traditionally, most clinicians apply some amount of pressure support during inspiration to compensate for the increased work of breathing related to artificial airway resistance. The amount of pressure support needed to counterbalance this resistance is highly variable, depending not only on the internal diameter of the endotracheal tube but also on flow, bend of the tube, and changing demands of the patient. This variability makes one level of pressure insufficient to meet these changing demands.[16]

Automatic tube compensation (ATC) compensates for endotracheal tube resistance via closed-loop control of calculated tracheal pressure. A ventilator with ATC compensates for the pressure drop across the endotracheal tube during inspiration by increasing the airway pressure and during expiration by decreasing airway pressure according to actual gas flow.[17–19] This technique uses a continuous calculation of the flow-dependent drop in pressure across the endotracheal tube. ATC is similar to PSV, but the pressure applied by the ventilator varies as a function of endotracheal tube resistance and flow demand. Most of the interest in ATC revolves around eliminating the imposed work of breathing during inspiration. During expiration, however, ATC may also compensate for that flow resistance by lowering the pressure in the expiratory limb transiently from its PEEP setting, helping reduce effective expiratory resistance and auto-PEEP.[17,20] In addition to overcoming the work of breathing imposed by the artificial airway, ATC may improve patient-ventilator synchrony by varying the flow commensurate with demand and may reduce air trapping by compensating for imposed expiratory resistance. During weaning trials, this technique may allow a more reliable prediction of patient performance when the tube is removed.

Indications for Mechanical Ventilation

Mechanical ventilation is instituted for a number of reasons (Table 9.1).[21] Most commonly, these indications are a combination of a failure to adequately oxygenate, ventilate, or meet the metabolic demands of a physiologically stressed patient. Clinical indicators such as tachycardia, arrhythmias, hypertension, and tachypnea, use of accessory respiratory muscles, diaphoresis, and cyanosis are used to diagnose respiratory distress. Type I respiratory failure is hypoxemic respiratory failure, defined as a partial pressure of oxygen in arterial blood (PaO_2) less than 60 mm Hg. Type II respiratory failure is hypercarbic respiratory failure, defined as $PaCO_2$ greater than 50 mm Hg, if elevated from patient baseline and associated with acidosis.[22] Blood pH is generally a better indicator than $PaCO_2$ for adjusting minute ventilation. Hypercapnia should not prompt aggressive intervention if pH remains acceptable and the patient remains alert. Hypercapnia is generally well tolerated, but this clearly depends on the underlying pathophysiology and comorbid conditions of the patient (e.g., right ventricular dysfunction). However, a sustained pH of 7.65 or greater or 7.10 or less is often considered sufficiently dangerous in itself to require control of minute ventilation by mechanical ventilation. Mechanical ventilation may also be instituted to maintain normal blood pH, decrease work of breathing, assist left ventricular function in the setting of acute decompensated heart failure, or for airway protection in the setting of toxic overdose, traumatic brain injury, or any other significant acute central nervous system illnesses. Box 9.1 suggests guidelines for setting basic operating parameters in a mechanical ventilator.

Box 9.1

Guidelines for the Initiation of Mechanical Ventilation

1. Choose the ventilator mode with which you are most familiar. The primary goals of ventilatory support are adequate oxygenation/ventilation, reduced work of breathing, synchrony between the patient and ventilator, and avoidance of high end-inspiratory alveolar pressures.

2. The initial FIO_2 (fraction of inspired oxygen) value should be 1.0. The FIO_2 thereafter can be titrated downward to maintain the SpO_2 (oxyhemoglobin saturation) at 92% to 94%. In severe acute respiratory distress syndrome (ARDS), >88% SpO_2 may be acceptable to minimize complications of mechanical ventilation.

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(VT) should be 8-10 mL/kg. Patients with acute respiratory failure (ARF) from e often require VT of 10-12 mL/kg to satisfy air hunger. In patients with ARDS, it is recommended to use a VT of 6 mL/kg and to keep inspiratory plateau pressure (IPP) 30 cm H_2O or less

4. Choose a respiratory rate and minute ventilation appropriate for the particular clinical requirements. Target pH, not $PaCO_2$. Initial respiratory rate is typically 10-12 breaths/min.

5. Use positive end-expiratory pressure (PEEP) in diffuse lung injury to support oxygenation and reduce the FIO_2.

6. In patients with COPD, avoid choosing settings that limit expiratory time and cause or worsen auto-PEEP.

7. When poor oxygenation, inadequate ventilation, or excessively high peak inspiration pressures are thought to be related to patient intolerance of ventilator settings and are not corrected by ventilator adjustment, consider initiating or increasing sedation or analgesia.

Adapted from Fundamental Critical Care Support, Des Plaines, IL, 2007, Society of Critical Care Medicine.

Table 9.1

Potential Indications for Mechanical Ventilation

Physiologic Mechanism	Clinical Assessment	Normal Range	Value(s)/Finding(s) Supporting Need for Mechanical Ventilation
Hypoxemia	$P(A-a)O_2$ gradient (mm Hg)	25-65	>350
	PaO_2/FIO_2 ratio	425-475	<300
	SaO_2	98%	<90% despite supplemental oxygen
Hypercarbia/inadequate alveolar ventilation	$PaCO_2$	35-45 mm Hg	Acute increase from patient's baseline pH <7.20 Mental status decline
Oxygen delivery/oxygen consumption imbalance	Elevated lactate	≤2.2 mg/dL	≥4 mg/dL despite adequate resuscitation
	Decreased mixed venous oxygen saturation	70%	<70% despite adequate acute resuscitation
Increased work of breathing	Minute ventilation	5-10 L/min	>15-20 L/min
	Dead space	0.15-0.30	≥0.5 (acute)
Inspiratory muscle weakness	NIP	80-100 cm H_2O	<20-30
	VC	60-75 mL/kg	<15-20
Acute decompensated heart failure	Jugular venous distention Pulmonary edema Decreased EF		Clinical judgment combined with the listed factors
Inadequate lung expansion	VT (mL/kg)	5-8	<4-5
	VC (mL/kg)	60-75	<10-15
	Respiratory rate (breaths/min)	12-20	≥35

A − a, alveolar-arterial; EF, ejection fraction; NIP, negative inspiratory pressure; VC, vital capacity; VT, tidal volume.

Mechanical Breath Generation

The breath generated by a mechanical ventilator can be separated into four *phases*: triggering, inspiration, cycling, expiration.

Triggering represents the change from expiration to inspiration and occurs either because of a drop in circuit pressure or diversion of flow (when patient triggered), or because of elapsed time. Sensitivity refers to a preset threshold of pressure or flow. When this threshold is reached, a mechanical breath is delivered. This threshold can be adjusted, and is usually set at −1 to −2 cm H_2O. If sensitivity is set too low, the ventilator will be triggered by any process that causes the airway pressure to drop below the set threshold. Such processes include patient motion, external compression, gastric suctioning, and air leaks in the circuit. Conversely, if the threshold is set too high, the work of breathing increases, as the patient must make a significant effort to d limit for inspiratory flow to occur. In the setting of pressure triggering, airway pressure t effort) in the proximal circuit, the expiratory valve closes, pressurization of the circuit occurs, and the patient receives a breath. Flow sensing was developed as an alternative to pressure triggering to reduce the delay in response time between neural input from the patient

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and delivery of gas volume by the ventilator.[23,24] In the setting of flow triggering, the patient's inspiratory effort induces a disruption of the constant flow in the ventilator inspiratory circuit. This change in flow signals the expiratory valve to close and for the ventilator to deliver the next breath. Flow triggering was initially demonstrated to decrease the work of breathing when compared to pressure triggering. However, with improvement in response time, monitoring, and feedback, pressure and flow triggering are similar in this regard.[25,26] Figure 9.2 is a representation of patient effort and ventilator response time.

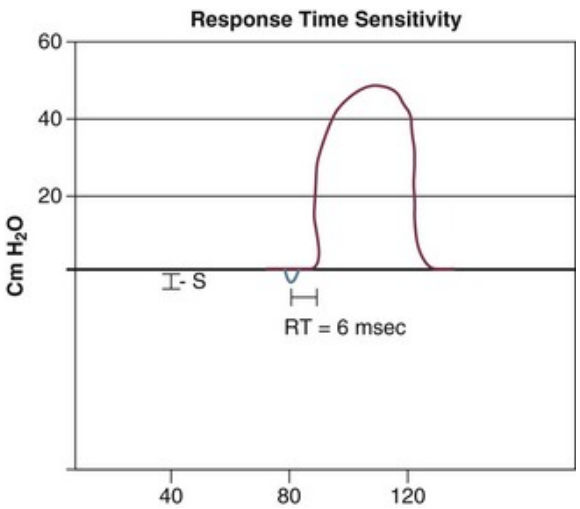
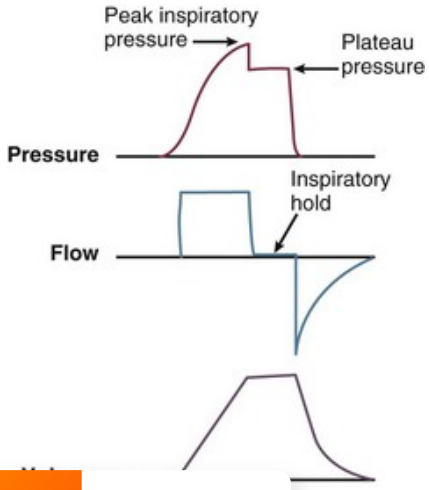


Figure 9.2 Pressure wave showing time relationship between patient inspiratory effort and ventilator response time (RT).

During *inspiration*, pressurized gas is channeled from the ventilator to the patient after the exhalation valve closes. This phase can be controlled by how one sets flow or pressure in the ventilator proximal to the open inspiratory valve. For example, volume-assist control is flow controlled and pressure-assist control is pressure controlled. Choice of control variable is largely the discretion of the clinician, as either can be manipulated to achieve set goals. It should be noted, though, that in volume-targeted ventilation, excessive airway pressures can arise secondary to worsening pulmonary mechanics. In this situation, the pressure alarm will cause a pressure limit to cycle to expiration. At the end of inspiration in volume-targeted ventilation, an inspiratory hold maneuver can be performed (Fig. 9.3), which can distinguish the peak airway pressure from the plateau pressure (because flow is stopped, resistance is negligible). In pressure-targeted ventilation, minute ventilation is not guaranteed. It is a function of the compliance and resistance of the respiratory system. The clinician therefore should monitor these physiologic changes closely to avoid untoward changes in either airway pressure or $PaCO_2$ levels.



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pressure measurement can be obtained in assist volume control mode by the performance of an inspiratory hold to better estimate the pressure in the lungs.

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With pressure control and pressure support (PS) breaths, the pattern of inspiratory flow is a natural decelerating pattern as the pressure gradient for flow decreases as pressure rises in the patient's lungs. The pattern of inspiratory flow with a flow-controlled breath is naturally square but can be computer altered to be *decelerating* or *sinusoidal* (Fig. 9.4). A square inspiratory flow pattern results in a rapid rise to a preset level (set by the clinician) followed by constant flow until cycling occurs. Decelerating flow results in a rapid rise to a maximum level followed by a gradual decrease until cycling. Sinusoidal flow pattern most closely represents normal physiologic breathing. It results in flow that gradually increases and then decreases during inspiration. The choice of inspiratory flow pattern should be based on patient characteristics, and a few common clinical scenarios should be familiar to the clinician. Square flow over time results in a shorter inspiratory time (I time), and therefore longer expiratory time (E time). For this reason it may be preferred in patients with obstructive physiology (chronic obstructive pulmonary disease [COPD] or asthma).[27,28] It is also usually tolerated better in patients with demand for high minute ventilation, such as severe metabolic acidosis or elevated ICP. In this situation, there is a potential for dyssynchrony to occur during the progression of the inspiratory phase if decelerating flow is chosen. The tradeoff is higher peak airway pressures with a square waveform, which may be ameliorated if it leads to a reduction in hyperinflation and iPEEP. Decelerating flow results in a longer I time and likely a better distribution of flow. With pulmonary pathophysiology involving heterogeneous distribution of injury (ALI as the prototype example), decelerating flow is likely the best choice, as the longer I time can lead to more homogeneous distribution of ventilation. The distribution of ventilation can be quite different when the flow pattern changes. A decelerating flow pattern yields the most even distribution under most abnormal airway conditions.[29] Studies have also demonstrated that a decelerating flow pattern improves the geographic distribution of lung vibration as a presumed surrogate of airflow.[30] The length of inspiration depends on several factors. In the pressure control mode of ventilation, the clinician directly controls the I time and I : E ratio. With pressure support the patient primarily controls I time. In flow-controlled (volume-targeted) ventilation, the I time is a function of set tidal volume (VT) and flow rate (VT/inspiratory flow rate) as well as flow wave characteristics (shape). The end-inspiratory alveolar pressure, as measured by an end-inspiratory hold, is the same for a given tidal volume regardless of the type of ventilator breath.

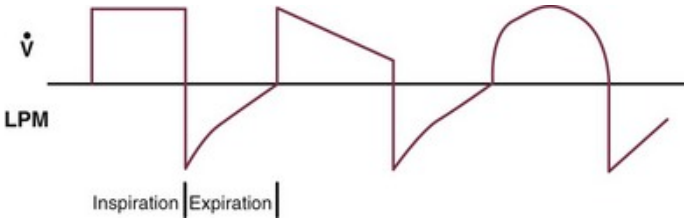

Figure 9.4 Depicted left to right are square, decelerating, and sine inspiratory flow waveforms as options for delivery of volume ventilation. Note that the square waveform produces the shortest inspiratory time and that the decelerating waveform does not return to zero flow at end inspiration. LPM, liters per minute; $\dot{V}$, flow.

Cycling is the transition from inspiration to expiration (closing inspiratory valve and opening expiratory valve). It can occur due to a threshold of decrease in flow, elapsed time, or delivered volume. *Flow-cycled breaths* are PS breaths. Because flow rate decreases dramatically as patient inspiratory effort decreases and then ceases, the patient exerts control not only of initiation of breath but also of its termination. When a predetermined decrement in flow occurs, inspiration is terminated and the patient cycles to expiration. This is typically set at 25% of initial flow, but can be manipulated based on different clinical conditions. For example, if a large leak occurs (bronchopleural fistula or ventilator circuit leak) it may be wise to set the cycle at a higher percentage of initial flow to terminate inspiration at the appropriate time. Strength and duration of patient inspiratory effort influence tidal volume. For safety reasons, PS breaths can be cycled due to excessive pressures (e.g., coughing during inspiration) or elapsed time (e.g., secondary to a leak in the system which would prolong inspiration). In *time-cycled breaths* (pressure control), inspiration is terminated after a preset interval, regardless of whether preset pressure has been achieved or a desired tidal volume has been delivered. Pressure is maintained at a preset level throughout inspiration, yielding a square pressure waveform. Pressure-targeted ventilation is time cycled, and the time can be adjusted to yield a precise I : E) ratio. In *volume-cycled breaths* (flow controlled), the clinician has selected the Volume is delivered until that volume is reached. Airway pressure is directly dependent the mechanical characteristics of the patient's respiratory system. For safety reasons, volume-cycled breaths will be pressure-cycled if airway pressures exceed the pressure alarm limit. In this

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situation, the delivered tidal volume will not reach the set volume target unless the pressure alarm limit is increased.

During expiration, flow from the ventilator is stopped, the exhalation valve opens, and gas is allowed out from the lungs. This release occurs passively. Inspiratory flow should cease prior to expiration. If this does not occur, the patient will develop auto-PEEP or intrinsic PEEP (iPEEP). This is most commonly seen in patients with expiratory flow limitation (COPD or asthma) or higher I : E ratios (see later).

Ventilator Modes

A ventilator mode describes ventilation over time and a set of specific combinations of breath characteristics delivered to a patient, including types of breaths, phase variables, and mandatory versus spontaneous breaths.[16] Clinician familiarity, unit, and institutional practice patterns determine to a large extent the mode that is employed. Also, data showing definitive improvement in clinically relevant outcomes when comparing one mode to another are lacking. Patient need and response to therapy should guide mode selection. Table 9.2 summarizes the features of some of the modes of mechanical ventilation, and Table 9.3 lists some of their advantages and disadvantages. Figure 9.5 also shows some characteristic waveforms of various modes of mechanical ventilation.

Table 9.2

Overview of Features of Selected Modes of Mechanical Ventilation

Ventilator Mode	Trigger	Control	Cycling	Inspiratory Flow
Continuous mandatory ventilation (CMV)	Time	Flow or pressure	Volume or time	Selected or decelerating
Volume control/assist control (VC/AC)	Patient or time	Flow	Volume	Square, decelerating, or sinusoidal
Pressure control/assist control (PC/AC)	Patient or time	Pressure	Time	Decelerating
Synchronized intermittent mandatory ventilation (SIMV)	Patient or time	Pressure for patient breaths Flow (VC) or pressure (PC) for ventilator breaths	Flow for spontaneous breaths Volume or time for ventilator breaths	Decelerating for spontaneous breath Square (VC), decelerating (VC or PC), sinusoidal for spontaneous breaths
Stand-alone pressure-support ventilation (PSV)	Patient	None	Flow	Decelerating

Table 9.3

Potential Advantages and Disadvantages of Selected Modes of Mechanical Ventilation

Mode	Advantage(s)	Disadvantage(s)
Controlled mechanical ventilation (CMV)	Rests muscles of respiration	Requires use of heavy sedation/ neuromuscular blockade
Assist volume control (AVC)	Reduced work of breathingGuarantees delivery of set tidal volume (unless peak pressure limit alarm is exceeded)	Potential adverse hemodynamic effectsMay lead to inappropriate hyperventilation and excessive inspiration pressures
Assist pressure control (APC)	Allows limitation of peak inspiratory pressures	Same as for AVCPotential hyperventilation or hypoventilation with lung resistance/ compliance changes
	Less interference with normal cardiovascular function	Increased work of breathing compared with assist controlPatient may find it difficult to adjust to two different types of ventilator breaths

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Stand-alone pressure-support ventilation (PSV)	Patient comfortImproved patient-ventilator interactionDecreased work of breathing	Apnea alarm is only backupVariable patient tolerance

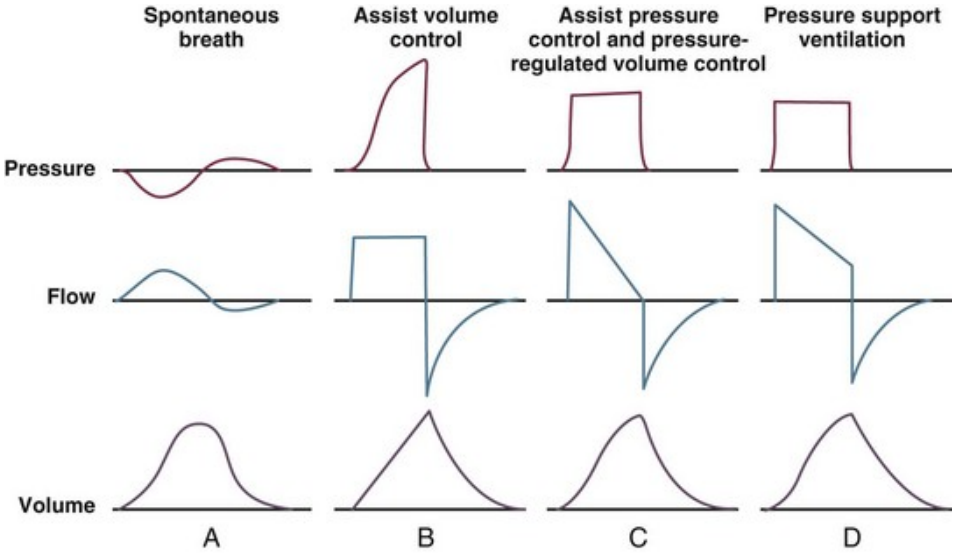

Figure 9.5 Characteristic pressure-flow waveforms with breathing spontaneously and various types of ventilation. **A,** Spontaneous breath. Such breaths are spontaneous, and inspiratory flow is achieved by the negative pressure generated by the respiratory muscles. Expiration occurs as these muscles relax. The combination of mandatory ventilator breaths (B or C) with spontaneous breaths (with or without pressure support) is called synchronized intermittent mandatory ventilation (SIMV). **B,** Assist volume control (AVC) ventilation. The flow is constant and pressure increases throughout inspiration. **C,** Assist pressure control (APC) ventilation or pressure-regulated volume control (PRVC). The pressure is constant and flow decreases throughout inspiration. In PRVC, the level of applied pressure may vary from one breath to the next. **D,** Pressure support ventilation. The pressure is constant and flow decreases throughout inspiration. When the flow reaches one fourth of its initial value, inspiration ends (flow-cycled). The flow and respiratory time are determined by patient effort and level of pressure support applied. The tidal volume varies from one breath to the next.

Continuous Mandatory Ventilation

With continuous mandatory ventilation (CMV), the patient has no influence on mechanical ventilation, and all breaths are mandatory. Patient-ventilator interaction is solely from the ventilator to the patient; it is time triggered and limited and cycled by the ventilator. Current ventilators do not have a CMV setting; this mode is essentially what is achieved when a patient on assist control (AC) ventilation has no interaction with the ventilator secondary to heavy sedation or neuromuscular blockade.

Assist Control Ventilation

In AC ventilation, a mandatory number of breaths are set and delivered with a set pressure or flow (assisted or unassisted), and if the patient's respiratory rate is higher than this backup setting (rate) additional assisted breaths to the preset pressure or flow are delivered. The target variable can be pressure (PC/AC) or volume (VC/AC).

Synchronized Intermittent Mandatory Ventilation

With synchronized intermittent mandatory ventilation (SIMV), the ventilator delivers a mandatory number of sure or flow (assisted or unassisted), similar to AC. However, spontaneous breaths are triggering during a timing window created around the delivery of mandatory breaths. reaths can be totally driven by patient effort or pressure-enhanced as pressure-controlled/flow-cycled (PS) breaths. The ventilator will attempt to synchronize the delivery of this mandatory

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breath with the spontaneous effort of the patient (if effort is present). If the patient does not trigger a breath, then the ventilator will deliver a regularly scheduled mandatory breath (time triggered).

Stand-Alone Pressure Support Ventilation

This mode of ventilation is patient triggered, pressure controlled, flow cycled, with a decelerating flow inspiratory waveform. Cycling to expiration typically occurs when flow rate decreases to a set percentage of inspiratory flow (typically 25% of initial inspiratory flow). However, if there is a leak in the system, inspiratory flow may never decrease to the cycle threshold. In this situation, for safety purposes, flow can be time cycled as well. During inspiration, the ventilator will adjust flow to maintain the preset pressure, and tidal volume is dependent on patient effort, as well as the mechanics of the respiratory system. Pressurization rate to the goal pressure is determined by the rise time, which can be adjusted on many ventilators. If the rise time is set too low, increased work of breathing can occur; if set too high, preset pressure may be exceeded (overshoot), causing early cycling to expiration. PSV is effective at decreasing patient effort and work of breathing.[31–33] Data suggest that most clinicians view PSV as a weaning mode of mechanical ventilation, despite its being fully capable of appropriate full support in patients with respiratory failure intact respiratory drive.[34]

Dual Control Modes

The preceding modes of mechanical ventilation are classically referred to as “conventional” ventilator modes, which is purely arbitrary. Dual control modes of ventilation manipulate pressure or volume on either a breath-to-breath basis or within the same breath. Breath-to-breath modes include volume support (VS) and pressure-regulated volume control (PRVC), also called “pressure control volume guarantee.” VS is PSV (pressure limited, flow cycled) that uses tidal volume as a feedback to adjust the pressure support level.[16] PRVC is pressure control ventilation (pressure limited, time cycled), but uses tidal volume as a conditional variable to adjust pressure to achieve desired tidal volume.

Other Modes of Mechanical Ventilation

Airway Pressure Release Ventilation

Airway pressure release ventilation (APRV) has been described in the literature since 1987.[35] It is a time-triggered, pressure-controlled, time-cycled mode of mechanical ventilation that exactly resembles AC/PC with very extended I : E ratios if the patient is not spontaneously breathing.[36,37] It has been described as continuous positive airway pressure (CPAP) with an intermittent time-cycled release phase to a set lower pressure. Whereas conventional modes of mechanical ventilation elevate airway pressure up from a set baseline to accomplish tidal ventilation, APRV employs very high I : E ratios to maintain mean airway pressure, and brief deflations to accomplish ventilation. When APRV is used in acute respiratory distress syndrome (ARDS) (primary use) short T_{low} is used whereas this mode can be used in patients without ARDS with longer T_{low} and is often called “bilevel positive airway pressure” (BiPAP) in that circumstance (Fig. 9.6). The expiratory valve in APRV also facilitates spontaneous breathing throughout the ventilatory cycle, facilitating ventilation. Because this mode is primarily used with ARDS with long T_{high} and very short T_{low}, breathing occurs primarily during T_{high}. Pressure high (P_{high}) is the baseline airway pressure that occupies most of the ventilatory cycle. Pressure low (P_{low}) is the release pressure and is set at 0 cm H_2O. Time high (T_{high}) is the length of time for which P_{high} is maintained and T_{low} is the length of time for which P_{low} is maintained. This is primarily semantics, but the term *APRV* implies longer I : E ratios than BiPAP, and a P_{low} setting of 0 at all times.

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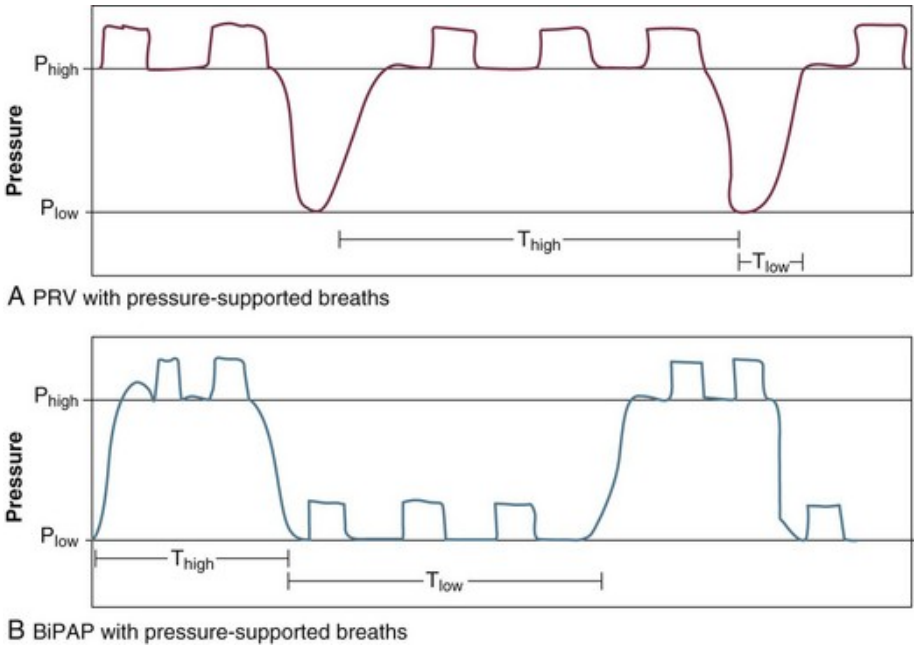

Figure 9.6 Pressure-time waveforms showing the use of airway pressure release ventilation (APRV). **A,** APRV with pressure-supported breaths. **B,** Bilevel positive airway pressure (BiPAP) with pressure-supported breaths.

Although outcome data are lacking compared to conventional ventilation using protective lung strategies, the reported advantages of APRV include sustained alveolar recruitment with improved oxygenation, higher mean airway pressures accomplished with lower Ppeak and Ppl, spontaneous breathing throughout the ventilatory cycle, and decreased use of sedation and neuromuscular blockade in severe ARDS.[37] The maintenance of spontaneous breathing may improve $\dot{V}/\dot{Q}$ matching by preferential ventilation of dependent lung regions, and the higher mean airway pressure, relative to Ppeak and Ppl, may limit VILI.

Alveolar recruitment is a pan-inspiratory phenomenon and alveoli that are recruited are more compliant than recruiting or nonrecruited alveoli. With prolonged elevated pressures, APRV likely recruits alveoli, which require a longer inflation with higher threshold opening pressures.[36,37] Sustained inflation maintains recruitment, decreases shunt and dead space, and employs the more compliant expiratory limb of the pressure-volume (PV) curve. Ventilation is determined by the stored kinetic energy at the high pressure, the intermittent release phase, and is augmented by spontaneous breathing. Although minute ventilation is decreased with this mode of ventilation, ventilation is also improved by a decrease in dead space.

Clinical data are limited but have shown improved oxygenation, less shunt and dead space, as well as decreased need for sedation and neuromuscular blockade.[37–41] Data on clinically relevant outcomes, such as mortality rates, mechanical ventilation days, and ICU days, are limited. This mode remains physiologically attractive for its effects on oxygenation and potential to limit VILI, but no specific recommendations can be made, given the lack of good outcome data.

High-Frequency Oscillatory Ventilation

High-frequency oscillatory ventilation (HFOV) is very different from conventional bulk flow ventilation and uses respiratory frequencies much higher and tidal volumes much lower than conventional modes. It is similar to APRV in that it aims to elevate mean airway pressure to maximize recruitment and oxygenation, while limiting Ppeak.[42] Oxygenated, humidified gas (bias flow) passes in front of an oscillating membrane and generates very small tidal volumes at very high respiratory rates. This is actively driven by a piston pump that oscillates oduces sinusoidal or somewhat erratic pressure waves that are actively driven in both on (unique in that expiration is not passively driven by elastic recoil). This component is rd movement of the diaphragm or piston of the oscillator. Resistance valves are used to apply a constant distending airway pressure, over which small tidal volumes are superimposed at a high

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respiratory frequency; in this fashion, it uncouples, for the most part, oxygenation and ventilation.

Given the fact the tidal volume is often less than anatomic dead space, HFOV relies on gas transport mechanisms much different from those employed by bulk flow ventilation. These mechanisms include some convective gas transport, but also molecular diffusion, pendelluft, coaxial flow, and Taylor dispersion.[43]

When initiating HFOV, the patient's endotracheal tube should be verified to be patent, as heavy secretions or kinks in the endotracheal tube will significantly harm ventilation. The mean airway pressure should be set at about 5 cm H_2O higher than that achieved with conventional settings. The bias flow delivers fresh gas into the ventilator circuit at 40 to 60 L/minute and helps maintain mean airway pressure.[43] The power control allows adjustment of amplitude (Δ pressure), which is set high enough to elicit vibrations of the patient to the lower abdomen or midthigh. Frequency helps determine ventilation and is set typically at 3 to 5 Hz. It should be noted, however, that at low frequencies, higher tidal volumes and bulk flow can be achieved, which assists in ventilation (and may contribute to VILI). Percentage of I time is set at 33% or 50%, depending on ventilation goals.

This effect would be predicted to be most beneficial when applied early in the course of severe ALI.[42,44] Recently completed clinical trials of the comparison of HFOV with ARDS net setting standard ventilation in patients at the onset of moderate or severe ARDS showed no benefit of HFOV, and one of the two trials showed increased mortality rate in the HFOV arm.[44,45] This would imply consideration of HFOV use only as salvage therapy in ARDS. The clinician should note that patients ventilated with HFOV will require heavy sedation or neuromuscular blockade.

Effort-Adapted Modes of Mechanical Ventilation

Early mechanical ventilators, such as the CMV mode setting, performed work on the patient and adapted the patient to the ventilator, with complete ventilator and physician control over breath characteristics and delivery. Although this can achieve adequate oxygenation and ventilation, it can come at a great expense to the patient, with side effects to include dyssynchrony, increased work of breathing, respiratory muscle weakness, and increased use of sedative infusions. Advances in ventilator technology, and increased recognition of the side effects of mechanical ventilation, have led to an increased awareness to adapt the ventilator to the patient, using the patient's physiology and demand to assist the respiratory muscles in proportion to effort and need. These effort-adapted modes of mechanical ventilation include proportional assist ventilation (PAV), neurally adjusted ventilatory assist (NAVA), and adaptive support ventilation (ASV).

PAV is a mode of partial support in which ventilator assist is delivered in proportion to patient effort. A defined level of assist, or unloading, relieves the resistive and elastic burden of the respiratory system.[46,47] The ventilator responds to the *mechanical output* of the patient and will amplify patient effort with a preset proportional amount of pressure support. The theory behind PAV is based on the equation of motion; based on changes in flow and volume, flow-proportional and volume-proportional pressure support is given. A percentage of effort is set by the clinician, and applied airway pressure develops as a function of volume to overcome elastance or a function of flow to overcome resistance.[46] Reported benefits of PAV include improved synchrony, better physiologic breathing pattern, and improved sleep quality.[48–54] A limitation of PAV is that it relies on the mechanical output from the patient, and therefore, continuous knowledge of the elastic and restrictive properties of the patient is a necessity. If PAV incorrectly estimates the mechanical properties, the ventilator may overassist, causing delayed inspiratory ending ("runaway phenomenon"). This problem has been improved by the development of PAV+ (PAV with load adjustable gain).

NAVA is similar to PAV in that it is also an effort-adapted mode of partial assist. Unlike PAV, which responds to the mechanical output from the patient, NAVA responds to the *neural input* from the electrical activity of the diaphragm (Edi). Pressure is applied in a linear proportion to Edi, and this requires the placement of an esophageal electrode (similar to nasogastric tube placement). The ventilator is triggered based on Edi or whichever comes first), therefore improving synchrony, as the time delay from patient brief. In addition, the amount of ventilator assistance varies based on Edi. As such, to ance, the patient must increase Edi. Reported benefits include improved synchrony[55–59] and preserved variability in breathing pattern.[46,60,61] High NAVA levels and high Edi may result in high

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inspiratory pressures and tidal volume delivery, especially in patients with unstable respiratory patterns or high respiratory drive.[62,63]

ASV ensures a set target minute ventilation based on measurements of the patient's E time constant and dynamic compliance, along with preset information of predicted body weight, minimum minute volume limit, and a pressure limit.[46] The ventilator attempts to minimize the work of breathing by combining the most effective combination of rate and tidal volume, while limiting pressure support. ASV potential benefits include decreased work of breathing and improved patient-ventilator interaction.

Effort-adapted modes of ventilator assist are physiologically attractive, especially considering that most conventional modes of mechanical ventilation are decades old. The decision to use these modes should be tailored to the individual patient. Patients with a high amount of dyssynchronous breaths and iPEEP secondary to obstructive physiology may benefit from a switch to these modes. They do not, however, ameliorate iPEEP, and extrinsic PEEP should be delivered appropriately. In patients who are severely hypoxemic or hemodynamically unstable, these modes should likely be avoided. All require an intact ventilator drive, and ongoing assessments of pulmonary mechanics is a necessity. Finally, the clinician must give up a significant amount of control with these modes of ventilation (which may be unappealing to some practitioners), and outcome data are currently lacking.

Positive End-Expiratory Pressure

CPAP maintains airway pressure above atmospheric pressure throughout the respiratory cycle by pressurization of the ventilator circuit. In mechanically ventilated patients, the purpose of CPAP is to achieve therapeutic PEEP in the presence of ALI or pulmonary edema (Fig. 9.7). In these patients PEEP serves to restore functional residual capacity (FRC), reduce IP shunt, shift ventilation to a more compliant portion of the PV curve, and prevent end-expiratory volume loss (derecruitment).[64] PEEP recruits previously nonaerated lung tissue and homogenizes regional distribution of tidal ventilation. The net effect on gas exchange reflects the balance between recruitment and overdistention. In the setting of obstructive physiology or expiratory flow limitation, PEEP serves less to improve oxygenation, but more to improve patient-ventilator synchrony and triggering.[27,28,65]

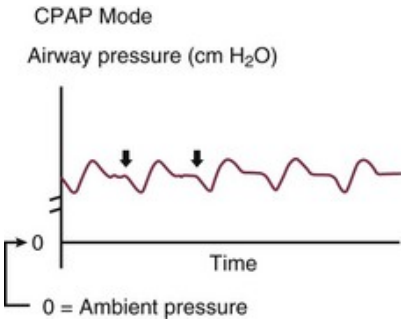

Figure 9.7 A pressure-time waveform in a patient with continuous positive airway pressure (CPAP) without application of pressure support ventilation is demonstrated. The patient is breathing spontaneously at an elevated baseline system pressure. With initiation of inspiration (*arrows*), pressure becomes more negative (but remains positive), and with expiration, pressure becomes more positive.

PEEP is not without side effects. Given the heterogeneous distribution of lung injury in ALI/ARDS, PEEP can overdistend more compliant lung units, contributing to VILI.[66] If PEEP leads to overdistention, it can augment dead space, increase pulmonary vascular resistance, and cause right-sided heart dysfunction. It can also decrease venous return and, in the setting of volume depletion, decrease cardiac output.

The optimal way to set PEEP is debated and controversial, as is the optimal level of PEEP to use.[67–82]

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etailed discussion of PEEP application in ARDS.

Monitoring the Ventilated Patient

Hemodynamics

Positive-pressure ventilation causes predictable physiologic changes and cardiopulmonary function is intimately linked—respiratory function alters cardiovascular function and vice versa.[83,84] The venous system is a low-pressure reservoir that contains about three fourths of our total blood volume.[83,85] This can be divided into the stressed and unstressed volume. The stressed volume contributes to the return of blood to the heart. The right ventricle's main function is to accept venous return and eject the optimal amount of blood into a (usually) highly compliant pulmonary vascular system.[84–87] This maintains a low right atrial pressure, thereby maximizing venous return and overall myocardial performance. A positive-pressure breath increases lung volume and increases intrathoracic pressure, which increases juxtacardiac pressure and therefore right atrial pressure. This can decrease venous return and therefore left ventricular preload several cardiac cycles later. At the same time, positive-pressure ventilation decreases left ventricular afterload by increasing the juxtacardiac pressure, therefore assisting ventricular contraction.

With respect to the mechanical ventilator, arterial blood pressure monitoring is commonplace in all intensive care units (ICUs). Though noninvasive cuff measurements can be adequate, invasive, continuous monitoring is much more informative when ventilator settings are dynamically changing. A fall in blood pressure temporally related to a ventilator change is a fairly specific indicator of a drop in cardiac output.[85] A narrow pulse pressure may indicate relative hypovolemia, although a widened pulse pressure may point to vasodilation or regurgitant cardiac valve disease. Pulse pressure variation (secondary to previously described heart-lung interactions) in mechanically ventilated patients is also the most accurate predictor of determining preload responsiveness.[88] Central venous pressure (CVP) is a reflection of right atrial pressure, and normally is low to maximize venous return. Despite the commonality of its use in determining volume status and preload responsiveness, data and physiology have shown that to be inaccurate.[89] This does not indicate the measurement of CVP is not of value. The absolute value and morphologic appearance (e.g., large v wave) of the CVP tracing can serve as valuable surrogates for right ventricular function, and an inspiratory fall in CVP can indicate preload responsiveness.[90] There are multiple other hemodynamic variables that could be measured in the mechanically ventilated patient, such as pulmonary artery occlusion pressure and mixed venous oxygenation, and these should be based on individual patient characteristics.

Pulse Oximetry

Pulse oximeters determine oxygen saturation by determining arterial blood light absorption at 660 nm and 940 nm wavelengths. The ratio of absorption of these two wavelengths is then calibrated against computer-stored algorithms to determine the oxygen saturation. Pulse oximeters are quite accurate at their upper range, but lose accuracy at lower oxygen saturations, require a good pulsatile signal, and have other limitations. These limitations include hypoperfusion, motion artifact, dyshemoglobinemias, ambient light, certain intravenous dyes, and deeply pigmented skin. Pulse oximetry is virtually ubiquitous in the ICU. Its use includes detection of hypoxemia, monitoring response to ventilator changes (such as PEEP setting), weaning FIO_2, and as a surrogate for pulmonary gas exchange.[91]

End-Tidal Capnography

End-tidal capnography ($EtCO_2$) is a useful way to monitor the concentration of exhaled carbon dioxide (CO_2), plotting it against time. A normal capnogram consists of a low value at baseline during inspiration and while emptying CO_2-free dead space. This is followed by a steep increase as alveolar units empty CO_2, and this increases progressively until a plateau is reached. A normal value (e.g., when ventilation and perfusion are matched) is about 5 mm Hg less than the $PaCO_2$, and a normal shape consists of a low baseline, steep [illegible] teau, and steep decline. The $EtCO_2/PaCO_2$ gap can widen in patients with altered [illegible] changes in cardiac output; the morphologic pattern of the waveform can be altered in patients with endobronchial intubation, expiratory flow limitation, and ventilator dyssynchrony.

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Ventilator Waveforms

Modern ventilators allow continuous monitoring of pressure, volume, and flow waveforms, all of which are plotted against time. They are useful in readily identifying the mode of mechanical ventilation being used, as well as identifying physiologic abnormalities. Examination of the flow waveform can allow detection of iPEEP. A ripple-like waveform can indicate water or secretion buildup in the ventilator circuit. A prolonged expiratory flow pattern can indicate expiratory flow limitation. The pressure waveform allows calculation of compliance of the respiratory system, and an estimation of transalveolar pressure with an end-inspiratory hold maneuver to calculate plateau pressure. An initial overshoot in the pressure waveform may signal high patient effort or an overshoot on ramp speed. An upward inflection at the end of the pressure waveform could signal overdistention, and a dip in an otherwise square pressure waveform can signal patient effort and dyssynchrony.

Maintaining Support of the Ventilated Patient

Sedation and Analgesia

Critically ill patients who are mechanically ventilated often require sedative and analgesic drugs, and pain is common among ICU patients.[92] The ideal sedative and analgesic regimen would provide adequate sedation and pain control, rapid onset of action, rapid recovery after discontinuation, minimal systemic accumulation, and minimal adverse effects—without raising health care costs.[93] The primary aim of these medications is to reduce the physiologic stress of respiratory failure and to improve the tolerance of mechanical ventilation. As more patients survive acute illness, the importance of goal-oriented sedation and analgesia has emerged, and the deleterious side effects of these medications is evident.[94]

Historically, mechanically ventilated patients have been treated with continuous infusions of benzodiazepines, opiates, and propofol.[95–97] Unfortunately, emerging data point to increased side effects associated with these medications, such as delirium, increased mechanical ventilation days, increased lengths of hospital stay, and higher mortality rate.[94,98,99] This has prompted interest in novel regimens with agents such as dexmedetomidine, an α_2-agonist with sedative and analgesic properties. Data have shown that dexmedetomidine use is associated with a reduction in delirium, improved cognitive function, and decrease in mechanical ventilation days.[100–104]

Regardless of the sedation regimen chosen, the mechanically ventilated patient should have sedation and analgesia titrated to a validated scale, such as the Richmond Agitation Sedation Scale, and delirium should be monitored at least daily, also with a validated scale (Confusion Assessment Method for the ICU [CAM-ICU]). All patients should be assessed daily for readiness to be liberated from the ventilator (spontaneous awakening trial [SAT]), and this should be paired with a spontaneous breathing trial (SBT).

Positioning

There is no optimal position for an individual patient and certainly no evidence-based gold standard. Predictable physiologic changes occur in patients when placed in the supine position, including reduced FRC, alteration in pleural pressure, reduced lung compliance, and change in $\dot{V}/\dot{Q}$ matching. The supine position should be avoided, as it is a risk factor for ventilator-associated pneumonia (VAP).[105,106] In patients with unilateral lung injury, the "good side" should be placed in the dependent position to optimize $\dot{V}/\dot{Q}$ match. Patients with severe hypoxemia (PaO_2:FIO_2 ratio < 100) should be considered for prone positioning early in the course of ALI, as this will likely improve oxygenation and may offer survival benefit in this patient cohort.[107]

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Secretion clearance is impaired in the critically ill patient, to increased secretion production and decreased mucociliary activity

and inability to cough effectively. Removal of oral and tracheobronchial secretions is commonplace and standard care of the mechanically ventilated patient. Because of insufficient data and complications, scheduled and routine suctioning should be avoided, but the decision is based on patient assessment. The use of closed-circuit catheters is now commonplace, but data are conflicting with regard to patient benefit when compared to open-circuit catheters. Prior to the suctioning procedure, the caregiver should be aware of the potential for complications, such as hypoxia, airway trauma, and cardiac arrhythmias.

Weaning From Mechanical Ventilation

Shortly after a patient is endotracheally intubated to initiate mechanical ventilation, thoughts should turn toward liberation from the ventilator. The withdrawal of mechanical support is a continuum from intubation until hospital discharge.[108,109] Depending on severity of illness, patient comorbid conditions, and critical care treatments needed, the timing of this may range from hours to weeks or months. There are several evidence-based guiding principles that shorten mechanical ventilation days across a broad cohort of critically ill patients.

The first step (and perhaps most important) in liberation from the ventilator is the targeted use of sedation, limitation of sedative and opioid infusions, and the monitoring for and aggressive treatment of delirium.[94] Patients should also undergo daily interruption of sedative medications (SAT), which has been shown to decrease ventilator days and ICU length of stay.[110] After awakening, the patient should be assessed for readiness to wean. The literature abounds with weaning predictors, all of which have modest sensitivity and specificity, including the frequency/tidal volume ratio.[109] Given this, as opposed to weaning predictors, the patient should undergo an SBT based on common sense, overall clinical trajectory, lack of hypoxia, and hemodynamic stability. The SAT should be paired with an SBT, as the SAT-SBT pairing decreases ventilator days, shortens ICU and hospital length of stay, and improves mortality rate.[111] An SBT can be conducted unassisted through a T-piece, on low-level PSV, or on CPAP, once or multiple times a day, and from 30 to 120 minutes; SIMV weaning is not recommended.[112–117] If a patient fails an SBT, a reason for that failure should be sought and corrected. This could include sedation, weakness, delirium, respiratory muscle fatigue, or left ventricular dysfunction. Also, after failing an SBT the patient should be placed immediately on comfortable full mechanical support, as delaying this and prolonging the SBT to the point of respiratory muscle exhaustion can delay extubation. After passing an SBT, the patient should be extubated unless a reason exists to leave the endotracheal tube in place (e.g., no cough reflex and heavy pulmonary secretions). The ability to predict successful extubation is challenging, and if the clinician never experiences a reintubation, he or she is likely not extubating patients based on the best available evidence.

Complications of Mechanical Ventilation

Hemodynamics

Blood pressure is simply the product of cardiac output and systemic vascular resistance (BP = CO × SVR). Although the transition of positive-pressure ventilation is associated with a predictable set of physiologic responses, the overall hemodynamic response is largely a consequence of underlying patient comorbid conditions (such as left ventricular dysfunction) and preload status. Immediately after endotracheal intubation, worsening hemodynamics are usually secondary to a decrease in preload, as right atrial pressure will increase, or a drop in arteriolar tone from sedation and analgesia. Any change in hemodynamics in the mechanically ventilated patient should prompt a thorough and almost algorithmic evaluation for potential causes.

Ventilator-Induced Lung Injury

Mechanical ventilation can be a lifesaving intervention. However, it has great potential for harm, and the [illegible] extend beyond normalization of gas exchange to providing safe and physiologically [illegible]tilation. Perhaps nowhere is this demonstrated more significantly than with the concept [illegible] with ALI do not die of hypoxia, but rather multiple organ dysfunction syndrome

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(MODS).[118–121] Applied airway pressures in patients with ALI distribute in a heterogeneous fashion, leaving more compliant lung units overdistended, and others collapsed and nonrecruited. Respiratory system compliance is related to the amount of normally aerated lung tissue that remains, the so-called baby lung.[122] This heterogeneity leads to a maldistribution of ventilation, with some alveoli overdistended, others collapsed throughout the respiratory cycle, and others cyclically opening and closing. VILI is a spectrum, classically defined as barotrauma, atelectrauma, and volutrauma (stretch injury).[123] All of these can lead to biotrauma, or the decompartmentalization of the inflammatory response secondary to increased alveolar epithelial-capillary endothelial permeability. This results in the release of biologic inflammatory mediators and the spread of injury to distant organs, causing MODS and death. VILI is much more complex than the preceding definitions and involves tissue stress and strain modifiers, complex molecular mechanisms, as well as gene activation and up-regulation.[124,125]

Human studies support the concept of VILI and the importance of a protective ventilation strategy with low tidal volumes and the use of PEEP. Low tidal volume ventilation is the only intervention shown to consistently improve outcome in ALI, and support for protective lung ventilation to decrease VILI from several well-conducted clinical trials exists.[71,72,126] This includes data showing a decrease in inflammatory mediators in patients ventilated with a protective lung strategy. In the absence of a patient-specific factor to suggest otherwise, a protective ventilation strategy, consisting of low tidal volume ventilation, PEEP setting, and limitation of plateau pressure, should be attempted at all times. Emerging data suggest that these strategies should be employed not only in patients with ALI, but in all patients receiving mechanical ventilation, especially those with known risk factors for the development of ALI.[127]

Ventilator-Associated Pneumonia

One of the most frequent complications of mechanical ventilation is the development of VAP.[128–139] VAP is discussed in Chapter 42.

Gastrointestinal Bleeding

Mechanical ventilation is a risk factor for gastrointestinal bleeding secondary to stress ulceration, trauma (especially traumatic brain injury), and major burns. Mechanical ventilation for longer than 48 hours is regarded as the most frequent risk factor.[140] The most effective treatment of stress ulceration is prevention. H_2-receptor antagonists and proton pump inhibitors have been shown to reduce the incidence of clinically important bleeding when compared to sucralfate and are considered the first-line therapy among many clinicians.[141–145]

Venous Thromboembolism

Venous thromboembolism (VTE) refers to the development of deep venous thrombosis (DVT) or pulmonary embolism (PE) in the critically ill patient. Patients in whom DVT develops have worse clinical outcomes, including prolonged mechanical ventilation.[146] Furthermore, VTE is common and frequently asymptomatic in mechanically ventilated patients. Given the adverse clinical outcomes associated with VTE, thromboprophylaxis with low-molecular-weight heparin or unfractionated heparin, both administered subcutaneously, should be given unless contraindicated.[147–149]

Noninvasive Positive-Pressure Ventilation

Noninvasive positive-pressure ventilation (NIPPV) offers the potential to provide ventilatory assistance without an invasive artificial airway. NIPPV may be accomplished using a facemask or nasal mask fitted to the face and connected through standard ventilator tubing to either a standard mechanical ventilator or smaller [illegible]fically to deliver noninvasive mechanical ventilation.

[illegible] and type II respiratory failure, NIPPV reduces dyspnea, decreases intubation rates, and improves mortality rates.[150–152] Similarly, in patients with cardiogenic pulmonary edema, NIPPV

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decreases intubation rates and improves survival.[152,153] NIPPV may also serve a critical role in decreasing mechanical ventilation days and improving weaning success, especially in patients with COPD recovering from type II respiratory failure.[154,155] Finally, immunocompromised patients, especially those with hematologic malignancies, should be considered for NIPPV.[156] Prior to initiation of NIPPV, the patient should be assessed for an adequate mental status for airway protection, hemodynamic stability, and lack of excessive secretions.

Key Points

• High-pressure alarms are triggered by patient factors (such as decreased compliance and increased resistance of the respiratory system) or by ventilator circuit malfunction (obstruction or kinking of the endotracheal tube).

• Low-pressure alarms are generally secondary to a leak in the system (ventilator circuit, endotracheal tube or cuff) or patient (large pressure loss from a bronchopleural fistula).

• Automatic tube compensation (ATC) compensates for endotracheal tube resistance via closed-loop control of calculated tracheal pressure.

• The breath generated by a mechanical ventilator can be separated into four phases: triggering, inspiration, cycling, expiration.

• With pressure control and pressure support breaths the pattern of inspiratory flow is a natural decelerating pattern as the pressure gradient for flow decreases as pressure rises in the patient's lungs.

• In AC ventilation, a mandatory number of breaths are set and delivered with a set pressure or flow (assisted or unassisted), and if the patient's respiratory rate is higher than this backup setting (rate) additional assisted breaths to the preset pressure or flow are delivered. The target variable can be pressure (PC/AC) or volume (VC/AC).

• With SIMV, the ventilator delivers a mandatory number of breaths with a set pressure or flow (assisted or unassisted), similar to AC ventilation. However, spontaneous breaths are delivered upon patient triggering during a timing window created around the delivery of mandatory breaths. These spontaneous breaths can be totally driven by patient effort or pressure enhanced as pressure-controlled/flow-cycled (PS) breaths.

• Although outcome data are lacking compared to conventional ventilation using protective lung strategies, the reported advantages of APRV include sustained alveolar recruitment with improved oxygenation, higher mean airway pressures accomplished with lower Ppeak and Ppl, spontaneous breathing throughout the ventilatory cycle, and decreased use of sedation and neuromuscular blockade in severe ARDS.[37]

• PAV is a mode of partial support in which ventilator assist is delivered in proportion to patient effort.

• NAVA is similar to PAV in that it is also an effort-adapted mode of partial assist. Unlike PAV, which responds to the mechanical output from the patient, NAVA responds to the neural input from the Edi. Pressure is applied in a linear proportion to Edi, and this requires the placement of an esophageal electrode (similar to nasogastric tube placement).

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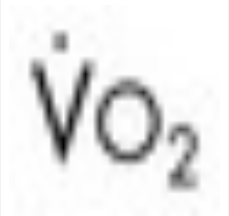

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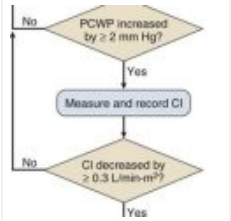

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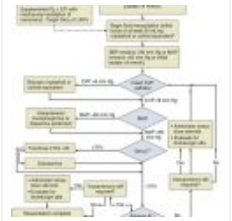

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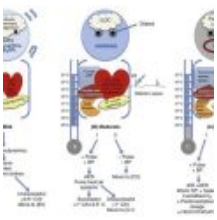

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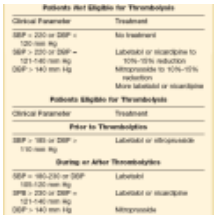

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